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UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

Ex parte PETER N. KAO, RONALD G. PEARL,
TOSHIHIKO NISHIMURA, and JOHN L. FAUL

Appeal 2008-5150
Application 10/801,729
Technology Center 1600

Decided:[1] April 29, 2009

Before DONALD E. ADAMS, RICHARD M. LEBOVITZ, and
MELANIE L. McCOLLUM, *Administrative Patent Judges.*

McCOLLUM, *Administrative Patent Judge.*

DECISION ON APPEAL

This is an appeal under 35 U.S.C. § 134 involving claims to treatment methods comprising administering an HMG-CoA reductase inhibitor. The

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[1] The two-month time period for filing an appeal or commencing a civil action, as recited in 37 C.F.R. § 1.304, begins to run from the decided date shown on this page of the decision. The time period does not run from the Mail Date (paper delivery) or Notification Date (electronic delivery).

Examiner has rejected the claims as indefinite, anticipated, and/or obvious. We have jurisdiction under 35 U.S.C. § 6(b). We affirm.

STATEMENT OF THE CASE

Claims 1, 3-8, 10, 12, 13, 19-24, 30, 32, 33, 37, and 39 are pending and on appeal (App. Br. 3). We will focus on claims 1, 3, 23, and 30, which read as follows:

1. A method of treating a lung proliferative vascular disorder in a patient comprising administering an HMG-CoA reductase inhibitor,

wherein the HMG-CoA reductase inhibitor is present in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, and which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient; and

wherein said lung proliferative vascular disorder is selected from the group consisting of primary pulmonary hypertension, secondary pulmonary hypertension, Eisenmenger's syndrome, chronic thromboembolic disease, pulmonary fibrosis, obliterative bronchiolitis, and lymphangioleiomyomatosis.

3. A method of treating primary pulmonary hypertension in a patient comprising:

administering an HMG-CoA reductase inhibitor in a pharmaceutical formulation comprising a pharmaceutically acceptable carrier at a dose of from about 0.1 to about 100 mg/kg per day, wherein the formulation further comprises in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, and which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient.

23. A method of treating primary pulmonary hypertension in a patient comprising:

administering an HMG-CoA reductase inhibitor in a pharmaceutical formulation comprising a pharmaceutically acceptable carrier suitable for pulmonary delivery at a dose of from about 0.1 to about 100 mg/kg per day, wherein the formulation further comprises in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, and which does

not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient;

wherein the HMG-CoA reductase inhibitor is administered by inhalation.

30. A method of reversing right ventricular hypertrophy in a patient suffering from pulmonary hypertension comprising administering an HMG-CoA reductase inhibitor.

Claims 1, 3-8, 10, 12, 13, and 19-24 stand rejected under 35 U.S.C. § 112, second paragraph (Ans. 4).

Claims 1, 3-8, 10, 12, 19-22, 30, 32, 33, and 39[2] stand rejected under 35 U.S.C. § 102(b) as anticipated by Liao (WO 00/56403 A1, Sep. 28, 2000) (Ans. 4).

Claims 13, 23, 24,[3] and 37[4] stand rejected under 35 U.S.C. § 103(a) as obvious over Liao (Ans. 5).

INDEFINTENESS

The Examiner finds that the "specification does not provide a standard for ascertaining the requisite degree of 'does not substantially increase...', and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention" (Ans. 4). In particular, the Examiner argues that "the meaning of the claims should be clear from the wording of the claim[s] alone" (*id.* at 7).

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[2] The Examiner's Answer also states that claim 38 is rejected on this basis. However, claim 38 has been cancelled (App. Br. 3).
[3] The Examiner's Answer states that claims 23-34 are rejected on this basis (Ans. 5). However, in view of the Final Rejection, it is clear that the Examiner intended to refer to claims 23-24 (Final Rej. 5; *see also* Ans. 14).
[4] The Examiner's Answer also states that claim 36 is rejected on this basis. However, claim 36 has been cancelled (App. Br. 3).

Appellants argue, however, that the phrase is explained in the Specification at page 10, lines 2-6 (App. Br. 6). "Appellants contend that it is well within the skill set of an ordinary practitioner to determine the NOS expression levels that are exhibited by normal healthy endothelial tissues" and that therefore one of ordinary skill in the art "would clearly understand what would constitute an enhancement of said expression levels above that exist[ing] in the healthy tissue. Accordingly, . . . the 'metes and bounds' of the claims would be clear to one of skill in the art." (*Id.*)

Issue

Did the Examiner err in concluding that the following phrase is indefinite: "which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient"?

Finding of Fact

1. The Specification states:

> The term "which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient" refers to an increase of NOS expression or activity to levels which would exist in normal healthy endothelial tissue, but not to any enhancement of NOS expression or activity above levels that would exist in normal healthy endothelial tissue.

(Spec. 10: 2-6.)

Principles of Law

"A claim is indefinite if its legal scope is not clear enough that a person of ordinary skill in the art could determine whether a particular composition infringes or not." *Geneva Pharms., Inc. v. GlaxoSmithKline*

PLC, 349 F.3d 1373, 1384 (Fed. Cir. 2003). "The standard of indefiniteness is somewhat high; a claim is not indefinite merely because its scope is not ascertainable from the face of the claims." *Amgen, Inc. v. Hoechst Marion Roussel, Inc.*, 314 F.3d 1313, 1342 (Fed. Cir. 2003). Rather, "[a] claim is indefinite if, when read in light of the specification, it does not reasonably apprise those skilled in the art of the scope of the invention." *Id.*

Analysis

The phrase at issue is defined in the present Specification (Finding of Fact (FF) 1). According to this definition, the phrase "refers to an increase of NOS expression or activity to levels which would exist in normal healthy endothelial tissue, but not to any enhancement of NOS expression or activity above levels that would exist in normal healthy endothelial tissue" (*id.*). Based on this definition, we find that a substantial increase refers to "any enhancement of NOS expression or activity above levels that would exist in normal healthy endothelial tissue" (*id.*). Thus, the phrase at issue encompasses increases that do not enhance "NOS expression or activity above levels that would exist in normal healthy endothelial tissue" (*id.*). Based on this definition, we agree with Appellants that the phrase is not indefinite.

Conclusion

The Examiner has not shown that the phrase at issue is indefinite. We therefore reverse the rejection under 35 U.S.C. § 112, second paragraph.

ANTICIPATION

The Examiner finds that "Liao teaches the use of HMG-CoA reductase inhibitor such as simvastatin in treating pulmonary arterial

hypertension . . . , wherein the simvastatin is administered in 0.01 mg/kg per day to 1000 mg/kg per day . . . , more preferably 50-500mg/kg" (Ans. 4).

> With respect to the activity of simvastatin . . . "in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, and which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient" (claim 1) . . . or "reversing right ventricular hypertrophy" (claim 30), [the Examiner deems] such properties or characteristics . . . to be inherent to the referenced method.

(*Id.* at 4-5.)

Issues

Did the Examiner err in concluding that Liao inherently discloses administering HMG-CoA reductase inhibitor in an amount that is effective to reduce vascular occlusion but does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient?

With regard to claim 3, did the Examiner err in concluding that Liao discloses administering an HMG-CoA reductase inhibitor to treat primary pulmonary hypertension?

With regard to claim 30, did the Examiner err in concluding that reversing right ventricular hypertrophy is inherent in the method disclosed in Liao?

Findings of Fact

2. The Specification discloses "a method of treating a lung proliferative vascular disorder in a patient . . . , which includes administering an antiproliferative agent[, a] preferred antiproliferative agent [being] a HMG-CoA reductase inhibitor" (Spec. 4: 16-18).

3. The Specification discloses that the "HMG-CoA reductase inhibitor is present in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, but does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient" (*id.* at 4: 18-21).

4. The Specification also discloses that the "HMG-CoA reductase inhibitor is generally administered in a dose of from about 0.1 to about 100 mg/kg per day, and more preferably from about 0.1 to about 20 mg/kg per day" (*id.* at 4: 27-28).

5. In addition, the Specification discloses that it was "discovered that administration of the antiproliferative agents causes reversal of the right ventricular hypertrophy associated with pulmonary hypertension" (*id.* at 10: 25-26).

6. Liao discloses the "use of HMG-CoA reductase inhibitors as upregulators of Type III endothelial cell Nitric Oxide Synthase" and "methods that employ HMG-CoA reductase inhibitors to treat conditions that result from the abnormally low expression and/or activity of endothelial cell Nitric Oxide Synthase in a subject" (Liao 1: 10-13).

7. In particular, Liao discloses that it is "known that individuals with pulmonary hypertension demonstrate reduced levels of Nitric Oxide Synthase expression in their pulmonary vessels and benefit clinically from inhalation of Nitric Oxide. The invention therefore is particularly useful for treating pulmonary hypertension." (*Id.* at 4: 13-16.)

8. Liao also discloses:

> The present invention, by causing an increase in ecNOS activity, permits not only the re-establishment of normal base-

> line levels of ecNOS activity, but also allows increasing such activity above normal base-line levels. Normal base-line levels are the amounts of activity in a normal control group, controlled for age and having no symptoms which would indicate alteration of endothelial cell Nitric Oxide Synthase activity.

(*Id.* at 10: 32 to 11: 4.)

9. In addition, Liao discloses:

> In abnormal circumstances, e.g. . . . pulmonary hypertension, etc., endothelial cell Nitric Oxide Synthase activity is depressed below normal levels. Surprisingly, when using the reductase inhibitors according to the invention, not only can normal base-line levels be restored in such abnormal conditions, but endothelial cell Nitric Oxide Synthase activity can be increased desirably far above normal base-line levels of endothelial cell Nitric Oxide Synthase activity. Thus, "increasing activity" means any increase in endothelial cell Nitric Oxide Synthase activity in the subject resulting from the treatment with reductase inhibitors according to the invention, including, but not limited to, such activity as would be sufficient to restore normal base-line levels and such activity as would be sufficient to elevate the activity above normal base-line levels.

(*Id.* at 11: 7-16.)

10. Liao also discloses:

> The reductase inhibitors are administered in effective amounts. In general, an effective amount is any amount that can cause an increase in Nitric Oxide Synthase activity in a desired tissue, and preferably in an amount sufficient to cause a favorable phenotypic change in the condition such as a lessening, alleviation or elimination of a symptom or of a condition.
>
> . . . Generally, doses of active compounds would be from about 0.01 mg/kg per day to 1000 mg/kg per day. It is expected that doses ranging from 50-500 mg/kg will be suitable.

(*Id.* at 19: 29 to 20: 7.)

11. In addition, Liao discloses treating relatively hypertensive mice, subcutaneously for 14 days, with 2 mg/kg simvastatin, an HMG-CoA reductase inhibitor (*id.* at 39: 14-19 & 14: 15-24).

Principles of Law

"[N]ot unlike a determination of infringement, a determination of anticipation, as well as obviousness, involves two steps. First is construing the claim, . . . followed by, in the case of anticipation or obviousness, a comparison of the construed claim to the prior art." *Key Pharms. Inc. v. Hercon Labs. Corp.*, 161 F.3d 709, 714 (Fed. Cir. 1998).

"A claim is anticipated only if each and every element as set forth in the claim is found, either expressly or inherently described, in a single prior art reference." *Verdegaal Bros., Inc. v. Union Oil Co.*, 814 F.2d 628, 631 (Fed. Cir. 1987). However, a disclosure that allows one skilled in the art to "at once envisage each member of [a] limited class" describes each member of the class "as if [the reference] had drawn each structural formula or had written each name." *In re Petering*, 301 F.2d 676, 681-82 (CCPA 1962).

"Newly discovered results of known processes directed to the same purpose are not patentable because such results are inherent." *Bristol-Myers Squibb Co. v. Ben Venue Labs., Inc.*, 246 F.3d 1368, 1376 (Fed. Cir. 2001).

> [I]t is elementary that the mere recitation of a newly discovered function or property, inherently possessed by things in the prior art, does not cause a claim drawn to those things to distinguish over the prior art. Additionally, where the Patent Office has reason to believe that a functional limitation asserted to be critical for establishing novelty in the claimed subject matter may, in fact, be an inherent characteristic of the prior art, it possesses the authority to require the applicant to prove that the

> subject matter shown to be in the prior art does not possess the characteristic relied on.

In re Best, 562 F.2d 1252, 1254-55 (CCPA 1977).

"Anticipation does not require the actual creation or reduction to practice of the prior art subject matter; anticipation requires only an enabling disclosure." *Schering Corp. v. Geneva Pharms.*, 339 F.3d 1373, 1380 (Fed. Cir. 2003). "[P]roof of efficacy is not required for a prior art reference to be enabling for purposes of anticipation." *Impax Labs. v. Aventis Pharms.*, 468 F.3d 1366, 1383 (Fed. Cir. 2006). "[T]he proper issue is whether the [prior art] is enabling in the sense that it describes the claimed invention sufficiently to enable a person of ordinary skill in the art to carry out the invention." *Id.*

Analysis

Claim 1 recites that the HMG-CoA reductase inhibitor is administered "in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, and which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient." We interpret this recitation to require that the inhibitor be administered in an amount that is effective to reduce vascular occlusion, but, as discussed above, does not enhance "NOS expression or activity above levels that would exist in normal healthy endothelial tissue" (FF 1).

Liao discloses doses of from about 0.01 mg/kg per day to 1000 mg/kg per day, particularly doses ranging from 50-500 mg/kg (FF 10). These ranges encompass or overlap the amounts disclosed in Appellants' Specification (0.1 to about 100 mg/kg per day) (FF 4). Thus, we conclude

that the Examiner has set forth a prima facie case that Liao discloses amounts that are effective to reduce vascular occlusion, but do not enhance NOS expression or activity above levels that would exist in normal healthy endothelial tissue (Ans. 4-5 & 9-10).

Appellants argue, however, that it is "clear that [Liao] intends a dose and route of administration of an HMG-CoA reductase that will result in increased ecNOS activity" (App. Br. 7). As a result, Appellants argue that Liao "teaches a method opposite of what is recited in the Applicants' claims" (*id.*).

We are not persuaded. As noted by Appellants, Liao discloses increasing ecNOS activity (FF 8). We recognize that Liao discloses increasing activity "sufficient to elevate the activity above normal base-line levels" (FF 9). However, Liao also discloses increasing activity "to restore normal base-line levels" (*id.*). In addition, Liao discloses that "individuals with pulmonary hypertension demonstrate reduced levels of Nitric Oxide Synthase expression in their pulmonary vessels" (FF 7). Thus, we agree that Liao discloses amounts that do not enhance "NOS expression or activity above levels that would exist in normal healthy endothelial tissue" (FF 1), as required by claim 1.

Appellants also argue that their "claimed invention is based partially upon the discovery that HMG-CoA reductase inhibitors show efficacy both in (1) preventing the development of smooth muscle cell hyperplasia (including medial hypertrophy), and in (2) inducing apoptosis in diseased and hypertrophied vascular tissues" and that this "discovery is in sharp contrast to methods, such as those disclosed in Liao, which teach the

administration of HMG-CoA reductase inhibitors to increase expression of ecNOS" (App. Br. 8). Accordingly, Appellants argue that "one of ordinary skill in the art could not practice the claimed invention without undue experimentation" (*id.*).

We are not persuaded. As discussed above, claim 1 does not exclude an increase in NOS expression or activity. Instead, it encompasses increases that do not enhance "NOS expression or activity above levels that would exist in normal healthy endothelial tissue" (FF 1). In addition, Liao discloses increasing activity "to restore normal base-line levels" (FF 9). Thus, we do not agree that one of ordinary skill in the art could not practice the method of claim 1 without undue experimentation. Instead, we agree with the Examiner that Liao "describes the claimed invention sufficiently to enable a person of ordinary skill in the art to carry out the invention." *Schering Corp. v. Geneva Pharms.*, 339 F.3d at 1380.

With regard to claim 3, Appellants argue:

> Liao *et al.* provide[s] the very general range of 0.01 mg/kg to 1000 mg/kg as being suitable for administration. However, the reference fails to provide any evidence that such a dose can be administered to an animal for the purpose of treating primary pulmonary hypertension. The examples provided by Liao *et al.* relate to cell culture assays, for example as illustrated in Figures 1-3, which show changes in ecNOS expression *in vitro*, or to cerebral infarction (Figures 4-6). There is no *in vivo* data provided by Liao *et al.* that would direct one of skill in the art in how to treat primary pulmonary hypertension by administering an HMG-CoA reductase inhibitor in a dose that increases ecNOS activity. While Liao *et al.* speculate[s] and assert[s] that this can be achieved; in fact there is no supporting evidence.

(App. Br. 9.)

We are not persuaded. Liao discloses that the invention "is particularly useful for treating pulmonary hypertension" (FF 7). Thus, we agree with the Examiner that Liao anticipates treating primary pulmonary hypertension. *See In re Petering*, 301 F.2d at 681-82 (a disclosure that allows one skilled in the art to "at once envisage each member of [a] limited class" describes each member of the class "as if [the reference] had . . . written each name"). In particular, we do not agree that Liao needs to provide *in vivo* data showing the treatment of primary pulmonary hypertension in order to anticipate claim 3. "Anticipation does not require the actual creation or reduction to practice of the prior art subject matter." *Schering Corp. v. Geneva Pharms.*, 339 F.3d at 1380.

With regard to claim 30, Appellants argue that Liao does not teach a method of reversing right ventricular hypertrophy in a patient suffering from pulmonary hypertension by administering an HMG-CoA reductase inhibitor (App. Br. 9). In particular, Appellants argue that "there is no mention or teaching of such a treatment" and that, "[b]efore the discovery reported in the instant application, it was highly unexpected that one could achieve an actual reversal of such a serious cardiac disorder" (*id.*).

We are not persuaded. The Examiner does not argue that Liao mentions reversing right ventricle hypertrophy. Instead, the Examiner argues that this effect is inherent in Liao's method. (Ans. 5.) We conclude that the Examiner has set forth a prima facie case that this effect is inherent in Liao's method and that Appellants have not rebutted the prima facie case.

Conclusion

Appellants have not shown that the Examiner erred in concluding that Liao inherently discloses administering HMG-CoA reductase inhibitor in an amount that is effective to reduce vascular occlusion but "does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient" and that Liao discloses administering an HMG-CoA reductase inhibitor to treat primary pulmonary hypertension. We therefore affirm the anticipation rejection of claims 1 and 3. Claims 4-8, 10, 12, and 19-22 have not been argued separately and therefore fall with claim 1. 37 C.F.R. § 41.37(c)(1)(vii).

In addition, Appellants have not shown that the Examiner erred in concluding that reversing right ventricular hypertrophy is inherent in the method disclosed in Liao. We therefore affirm the anticipation rejection of claim 30. Claims 32, 33, and 39 have not been argued separately and therefore fall with claim 30. 37 C.F.R. § 41.37(c)(1)(vii).

OBVIOUSNESS

The Examiner finds that "Liao teaches the use of HMG-CoA reductase inhibitor such as simvastatin in treating pulmonary arterial hypertension . . . , wherein the simvastatin is administered . . . in various dosage forms including oral, rectal, topical, nasal, interdermal or parenteral" (Ans. 4). The Examiner concludes:

> [T]hose of ordinary skill in the art would have . . . readily optimized effective delivery forms as determined by good medical practice and the clinical condition of the individual patient. Determination of the appropriate delivery dosage forms for treatment involving each of the above mentioned formulations is routinely made by those of ordinary skill in the

> art and is within the ability of tasks routinely performed by them without undue experimentation, especially in light of the conventional drug delivery forms known in pulmonary hypertension treatment of art.

(Ans. 5-6.)

Issue

Did the Examiner err in concluding that it would have been obvious to administer the HMG-CoA reductase inhibitor by inhalation?

Finding of Fact

12. Liao discloses:

> A variety of administration routes are available. The particular mode selected will depend, of course, upon the particular drug selected, the severity of the condition being treated and the dosage required for therapeutic efficacy. The methods of the invention, generally speaking, may be practiced using any mode of administration that is medically acceptable, meaning any mode that produces effective levels of the active compounds without causing clinically unacceptable adverse effects. Such modes of administration include oral, rectal, topical, nasal, interdermal, or parenteral routes.

(Liao 21: 3-9.)

Principle of Law

The test of obviousness is "whether the teachings of the prior art, taken as a whole, would have made obvious the claimed invention." *In re Gorman*, 933 F.2d 982, 986 (Fed. Cir. 1991).

Analysis

Liao discloses administering the HMG-CoA reductase inhibitor "using any mode of administration that is medically acceptable, meaning any mode that produces effective levels of the active compounds without causing

clinically unacceptable adverse effects," and that "[s]uch modes of administration include oral, rectal, topical, nasal, interdermal, or parenteral routes" (FF 12). In addition, Liao acknowledges that inhalation is a known mode for administering an agent for treating pulmonary hypertension (FF 7). Thus, we agree with the Examiner that it would have been obvious to administer the HMG-CoA reductase inhibitor by inhalation.

Appellants argue that Liao "lacks specific guidelines for dose and route of administration whereby one could accomplish the sought after result," i.e., treating primary pulmonary hypertension (App. Br. 11). In addition, Appellants argue:

> In view of this lack of teaching by Liao *et al.* it is not obvious that one should "optimize" effective delivery forms. The process of optimization lacks a credible foundation when the desired result has not yet been obtained, and becomes, instead, a searching for hope of success, without a reasonable expectation that such will be found.

(*Id.*)

We are not persuaded. As discussed above, we agree with the Examiner that Liao discloses treating primary pulmonary hypertension. Liao also discloses dosage amounts (FF 10). Thus, we agree with the Examiner that it would have been obvious to administer the recited dosage amounts of the HMG-CoA reductase inhibitor by a mode of administration known for use in treating pulmonary hypertension, i.e., inhalation (FF 7).

Conclusion

Appellants have not shown that the Examiner erred in concluding that it would have been obvious to administer the HMG-CoA reductase inhibitor by inhalation. We therefore affirm the obviousness rejection of claim 23.

Claims 13, 24, and 37 have not been argued separately and therefore fall with claim 23. 37 C.F.R. § 41.37(c)(1)(vii).

SUMMARY

We affirm the anticipation rejection of claims 1, 3-8, 10, 12, 19-22, 30, 32, 33, and 39 and the obviousness rejection of claims 13, 23, 24, and 37. However, we reverse the rejection under 35 U.S.C. § 112, second paragraph.

TIME PERIOD FOR RESPONSE

No time period for taking any subsequent action in connection with this appeal may be extended under 37 C.F.R. § 1.136(a).

AFFIRMED

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